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Hashimoto´S Encephalopathy: Diagnosis by Exclusion in an Atypical Case

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Abstract | **Case Report**

Hashimoto's encephalopathy is a rare disease with an estimated prevalence of 2.1 cases per 100,000 people, due to its nonspecific clinical presentation, a predilection for the female sex, and an age group between the fourth and fifth decades of life. This pathology describes a variety of brain disorders that result in altered brain function, manifesting as cognitive impairment, behavioral changes, memory problems, and even neurological symptoms. For a correct diagnosis, we must have a clinical suspicion based on sex, age group, clinical presentation with neurological alterations without etiology that meet the six criteria, include laboratory and imaging studies to rule out infectious causes, anatomical, functional, and hormonal alterations, with the search for specific antibodies (anti-Tg and anti-TPO) against this autoimmune thyroid disease, making use of special imaging studies such as plain and contrast cranial MRI. The mainstay of treatment for this condition is steroids, which have a good response; however, it presents a diagnostic and therapeutic challenge in our setting due to its low prevalence and underdiagnosed cases, with a very low number of reported cases.

Keywords: Hashimoto's encephalopathy, autoimmune, antibodies, steroids, neurological.

INTRODUCTION

Encephalopathy is a broad term used to describe a variety of brain disorders that result in altered brain function. It can manifest as cognitive impairment, behavioral changes, memory problems, and even neurological symptoms [1]. ashimoto's encephalopathy (HE), also known as steroid-responsive encephalopathy associated with autoimmune thyroiditis (ESEATA), is a neurological syndrome associated with elevated serum levels of antithyroid antibodies, with preserved or altered thyroid function, and which usually responds to treatment with glucocorticoids. Its etiology is still unknown; the available evidence stipulates autoimmunity due to vasculitis or another inflammatory process [2-7]. Epidemiologically, it is a rare condition, with an estimated prevalence of 2.1 cases per 100,000 people. It is more common in women aged 40 years or older, particularly those with a family history of autoimmunity and thyroid disorders such as Hashimoto's thyroiditis [6,8]. The condition has been reported in different parts of the world, and its incidence may vary depending on geographic and demographic location. There are no reports of its prevalence in Mexico.

The clinical picture often presents with a wide range of symptoms that may be sudden or insidious in onset and may include: confusion, seizures, mild cognitive impairment, altered level of consciousness, focal symptoms similar to a stroke, hallucinations, transient global amnesia, and psychiatric manifestations.

The diagnosis for Hashimoto's encephalopathy can be made when the following six criteria are met: encephalopathy with seizures, myoclonus, hallucinations or stroke-like episodes, overt subclinical or mild thyroid disease (usually hypothyroidism), normal or nonspecific abnormal brain MRI, presence of serum thyroid anti-thyroid peroxidase, anti-thyroglobulin antibodies, absence of well-characterized neuronal antibodies in serum and CSF, reasonable exclusion of alternative causes [5].

The prognosis for Hashimoto's encephalopathy (HE) can vary widely depending on several factors, such as antibody levels, the severity of symptoms themselves, the speed of diagnosis, and the effectiveness of treatment. In 90% of cases with Hashimoto's encephalopathy, a significant number of people with HE

may experience improvement in their symptoms and quality of life, and up to 20% may be left with sequelae [9,10].

Clinical Case

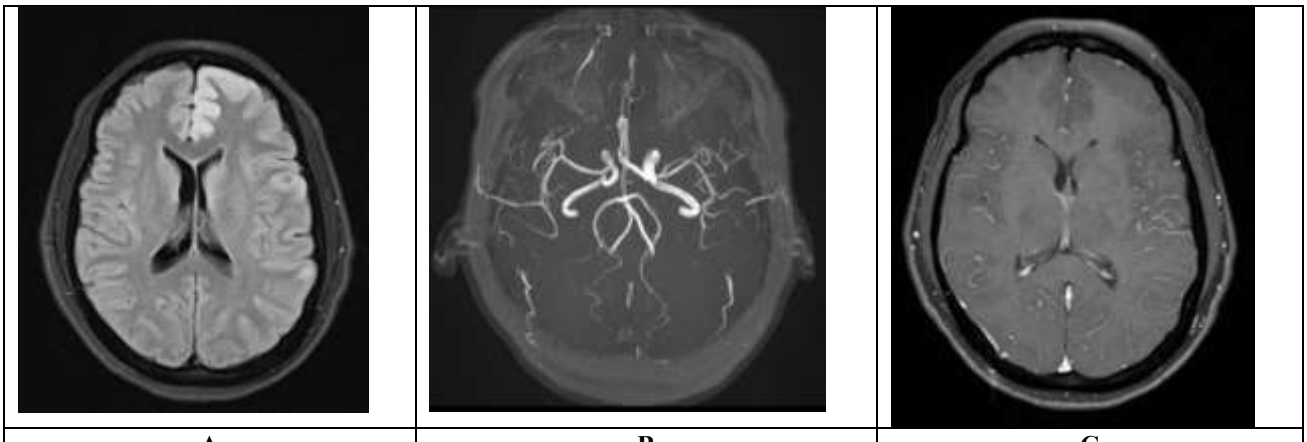
In the following case, we address a 39-year-old woman with no past medical, non-medical, or familial history. She presented symptoms of depression approximately six months ago and self-medicated with "Phentermine" for four months to lose weight. She began with a daytime holocranial headache with a visual analog scale of 7/10, followed by a generalized tonic-clonic seizure lasting less than a minute, with sequelae of right hemiplegia, dysarthria, mixed aphasia, drowsiness, and neck stiffness. A simple cranial computed tomography was performed, which was reported to be normal, and a lumbar puncture reported normal cerebrospinal fluid with a rock water appearance: glucose: 77 g/dL, protein: 38 g/dL, and no leukocytes. Empirical treatment was started with ceftriaxone, vancomycin and dexamethasone, showing minimal improvement in the following 48 hours, so the diagnostic approach was expanded in search of infectious causes, anatomical, structural, hormonal alterations and autoimmune disease.

Thyroid hormones were requested (Table 1), as well as anti-HIV antibodies: negative, anti-hepatitis C: negative, anti-hepatitis B surface antibody: negative. CSF culture was reported without development at 72 and 168 hours, with negative India ink, and no bacteria were observed in the Gram stain. The real-time polymerase chain reaction report for herpes simplex virus 1,2, Epstein-Barr, Varicella zoster, Parechovirus, Enterovirus, Toscana virus, L. monocytogenes, N. meningitidis, H. influenzae, S. agalactiae, C. neoformans, T. pallidum, C. Burnett and B. burgdorferi reported: non-reactive (negative); with this, infectious and hormonal causes were excluded, so the approach began in search of anatomical or structural alterations in the central nervous system and autoimmune disease.

Table 1: Thyroid profile

Bookmarks	July 2024	January 2025	Reference value
TSH	0.5688 uIU/ml	1.9039 uIU/ml	0.35-4.94 uIU/ml
T4	5.69 ug/dl	7.86 ug/dl	4.5-12.5 ug/dl
T3	0.64 ng/ml	0.78 ng/ml	0.64-1.52 ng/ml
FT3	1.97 pg/ml	2.40 pg/ml	1.8-4.2 pg/ml
FT4	1.03 ng/dl	1.12 ng/dl	0.89-1.76 ng/dl

An MRI was subsequently performed (Image 1) in search of anatomical and microvascular alterations at the level of the central nervous system, which could provide the etiology of the condition. Serum immunoglobulin studies were requested, type IgG: 3765 mg/dl, IgA; 120 mg/dl, IgM 146 mg/dl, Complement C3 81 mg/dl, C4 5.3 mg/dl, rheumatoid factor 20.8 U/ml and specific antibodies (Table 2) in search of an autoimmune cause associated with the thyroid.



A **B** **C**

Image 1: Simple and contrast-enhanced magnetic resonance imaging of the skull

A. Flair: Signal intensity changes in gray matter, predominantly in the frontal area, as well as in some parietotemporal regions on the left side. Asymmetry of the lateral ventricles, with a lower amplitude on the left side compared to the contralateral side.
B. Angiographic sequence: tortuosity of the internal carotid arteries, predominantly right cavernous segment, hypoplasia/agenesis of the anterior cerebral artery, focal area of decreased diameter of the left anterior cerebral artery, segment A2.
C. Leptomeningeal enhancement, without associated artifacts.

Table 2: Antibody profile

Name	Result	Reference value
Antinuclear antibodies by immunofluorescence	Negative	Positive titer >1:80/ negative
Anti-Ro	Negative	Positive >25 IU/ml/ negative
Anti-thyroglobulin (anti-Tg) antibodies	95.97 IU/ml	<100 IU/ml
Antithyroid peroxidase antibodies (anti-TPO):	141.11 IU/ml	< 15 IU/ml

As a complement, an electroencephalogram was requested, which reported sleep-wake activity with mild generalized dysfunction of brain electrical activity.

DISCUSSION

In retrospect, due to the partial improvement shown by the steroid (dexamethasone) that had been initially administered for suspected neuroinfection, the absence of infection at the systemic level as well as in the CSF, elevated antithyroid antibodies, and no abnormal thyroid hormones, a diagnosis of "Hashimoto's encephalopathy" was made by exclusion, and treatment was started with methylprednisolone 1 gram every 12 hours and human immunoglobulin 30 grams per day for 5 days; at the end, rituximab 1 g was continued as a single dose. The patient remained hospitalized for 20 days with recovery of motor function, and did not present any new seizures or deterioration of functional status. She was discharged with oral steroid therapy for monitoring by the outpatient clinic. Follow-up was carried out over the next six months with a thyroid profile (Table 1), which was found to be normal. The patient continued with clinical improvement, without presenting any further exacerbation of the condition.

CONCLUSIONS

Although Hashimoto's encephalopathy is a rare disease, it is part of the autoimmune thyroid diseases (ETA) with an incidence of 5% in the world's population. Therefore, knowing the clinical presentation, age group, sex predilection, and the diagnostic and therapeutic approach will allow us to successfully treat the patient. In the previously discussed case, the approach was initially neurological; all infectious causes, anatomical structural variants, and metabolic causes were initially ruled out; The diagnostic pillars were the age group of the fourth decade of life, female sex, insidious clinical presentation, as well as the previous response to steroid treatment that she had had empirically, in addition to timely support in the specific antibodies of anti-TPO, which has a sensitivity of 81.5%, specificity of 96% and anti-Tg, which has a sensitivity of 52.5%, specificity of 94%, high-resolution imaging studies such as simple magnetic resonance, contrast-enhanced, with Flair, as well as electroencephalogram; with this series of studies we were able to establish the precise diagnosis of Hashimoto's encephalopathy. Despite having a nonspecific clinical picture, the approach was a diagnostic challenge. All resources were used to reach a timely diagnosis and provide the specific treatment, which in this case is with steroid therapy initially in cycles, then orally, obtaining a favorable response, with recovery of the functional state.

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